Appendix P

Newmark Project Preliminary Baseline Risk Assessment

TABLE OF CONTENTS

TABLE OF CONTENTS (Cont'd.)

LIST OF TABLES

FOREWORD

This document presents the results of the preliminary baseline risk assessment for the Newmark Operable Unit (OU) of the Newmark Superfund Project. The purpose of this preliminary risk assessment is to support the selection of an interim remedial action. Interim remedial actions do not require a completed baseline risk assessment, although enough information must be available to demonstrate that action is necessary to stabilize the site, prevent further degradation, or achieve significant risk reduction quickly (Preamble to the NCP Final Rule, Federal Register, Vol. 55, 8704).

The preliminary risk assessment estimates potential, not actual, risk. The risk estimates are based on the unlikely assumption that federal and state drinking water standards (e.g., Maximum Contaminant Levels or MCLs) are not enforced, in which case residents of the San Bernardino area could be served contaminated groundwater extracted from within or near the OU area of contamination. This is only an assumption; groundwater served to consumers is currently believed to satisfy all enforceable drinking water standards.

The preliminary risk assessment examines exposure to VOCs detected in groundwater in the Newmark OU area. Vadose zone remediation is not a goal of the interim action, therefore, exposure to contaminated soil or soil gas will not be addressed in this preliminary risk assessment. The possibility of exposure to contaminants in the unsaturated zone was explored by sampling for contamination in soil at the "CAT pit" (see RI/FS Section 5.1.1) and by a screening study of the air in nearby residences (see Appendix K). None of the results indicated a measurable exposure from the soil gas pathway.

BACKGROUND OF THE BASELINE RISK ASSESSMENT

Baseline risk assessments are conducted at Superfund sites to fulfill one of the requirements of the National Contingency Plan (NCP). The NCP (40 CFR Part 300) is a document that sets forth the manner in which Superfund remediations are to be planned and conducted. The NCP requires development of a baseline risk assessment at sites listed on the National Priorities List (NPL) under CERCLA, as amended by SARA. The CERCLA process for baseline risk assessments is intended to address both

human health and the environment. However, due to the highly urbanized setting of the Newmark OU, the focus of the baseline risk assessment presented in this section is on human health issues, rather than environmental issues.

In general, the process of risk assessment involves the qualitative or quantitative characterization of potential health effects of specific chemicals on individuals or populations. This process comprises four basic steps (NCP): 1) hazard identification, 2) dose-response or toxicity assessment, 3) exposure assessment, and 4) risk characterization. The purpose of each element is summarized below. The risk assessment is organized as follows:

- **Identification of Chemicals of Potential Concern (COPC)**: Identifies the chemicals evaluated in the assessment.

- **Exposure Assessment**: Identifies potential pathways by which exposures could occur; characterizes the potentially exposed populations; and estimates the magnitude, frequency, and duration of exposure.

- **Toxicity Assessment**: Summarizes the toxicity of the chemicals of concern and the relationship between magnitude of exposure and adverse effects.

- **Health Risk Characterization**: Integrates the toxicity and exposure assessments to estimate the potential risks to public health and the environment from exposure to site chemicals.

- **Uncertainties**: Summarizes limitations of the data and methodology used in the risk assessment.

- **Ecological Assessment**: Qualitatively evaluates areas where potential ecological impacts may occur.

1.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN (COPC)

The preliminary risk assessment is based on groundwater data collected during EPA sampling in the Newmark OU area during 1992. Data from both EPA monitoring wells and previously existing non-EPA water supply wells and monitoring wells were included. The similarity of VOC levels in each group supported combining the data for the analysis. Groundwater sampling results from this effort are presented in RI/FS Tables 5-3 through 5-5. The analytical data were analyzed and validated in accordance with EPA CLP procedures (EPA, 1991a). Results (noted by a "J" qualifier) which fell between the instrument detection limit (IDL) and the contract required detection limit (CRDL) are considered estimates of limited usefulness.

Thirteen VOCs were identified in groundwater from the OU area, although only three (PCE, TCE and 1,2 DCE) were detected at levels that could be used without qualification (see Table 1). PCE was the most prevalent VOC, and the detection of PCE in a well was used to determine whether that well was included within the contaminant plume of the Newmark OU. PCE was detected in 26 wells (11 EPA monitoring wells and 15 existing wells) within the area considered for the Newmark OU, although levels were too low for accurate quantification of PCE in 7 of these wells. Monitoring well 01 (WMW01) did not contain detectable levels of any VOCs except toluene and butene (both below the quantifiable detection limit). This well has not been included in the evaluation of groundwater exposures. Since neither toluene nor butene were detected in any other well, these compounds are not considered in the preliminary risk assessment. Two of the remaining eleven compounds (dichlorofluoromethane and trichlorofluoromethane) were only tentatively identified, and are not considered further. The nine remaining chemicals detected in groundwater have been chosen as COPCs for the preliminary risk assessment. Six of these compounds were detected at levels too low for quantification, and were not detected at all in many of the wells.

Inorganic compounds (e.g., metals) were detected in EPA monitoring wells at levels consistent with newly constructed wells (see RI/FS Table 5-4). In operating public water supply wells within the Newmark OU, long-term monitoring required by health officials have detected no inorganic chemicals

exceeding background levels for the area and none of public health concern. These chemicals are not included as COPC in the baseline risk assessment.

2.0 EXPOSURE ASSESSMENT

Exposure assessment is the determination or estimation of the exposure magnitude, frequency, duration, and route. This section identifies the potentially exposed human populations and the means by which these individuals can come into contact with contaminants detected in groundwater in the OU area.

2.1 POTENTIALLY EXPOSED POPULATIONS

Most of the land in the OU area is developed for residential, commercial, and industrial land use. Water supply wells in the highly contaminated portions of the OU area have been shut down or, in three locations, had treatment installed. For the purpose of this preliminary risk assessment, **it is assumed that future uses of the groundwater in the OU area would include use of untreated groundwater for domestic purposes**. The potentially exposed populations include residents and workers in the OU area.

2.2 POTENTIAL EXPOSURE PATHWAYS

An exposure pathway is the route by which a receptor makes contact with a contaminant source. This preliminary risk assessment is limited to the groundwater exposure pathway. Exposure to contaminants in groundwater could occur through the use of groundwater for domestic purposes. In residences, people can be exposed to contaminants from ingestion of water used for drinking and cooking. They can also be exposed through dermal absorption of contaminants, primarily during bathing and showering, and inhalation of VOCs released from the water into the household air during showering, bathing, cooking or by the use of household appliances such as washing machines. Exposure to contaminants in

groundwater can also occur through the use of groundwater for industrial purposes. Workers could be exposed through dermal absorption of contaminants or inhalation of VOCs.

Residents and workers could also be exposed to contaminants in groundwater through the transport of VOCs from groundwater through soil and into ambient air or into the foundation of a building. This is unlikely to be a significant exposure route in the OU area because the depth to groundwater throughout the area is greater than 100 feet. Results of an EPA screening study of the air quality of residences in the Newmark OU support this conclusion.

Groundwater does not discharge to surface water in the Newmark OU area.

This preliminary risk assessment will quantitatively address risk due to exposure to contaminants in groundwater used for domestic purposes. Based on potential exposure frequency, duration, and estimated intake exposures, residents exposed to contaminated groundwater used for domestic purpose are expected to be the maximally exposed population.

2.3 QUANTIFICATION OF EXPOSURE

Exposure is defined as the contact of an organism with a chemical or physical agent. The measure of exposure (or intake) is expressed as milligrams of chemical per kilogram of body weight per day (mg/kg/-day). Six basic factors are used to estimate intake: exposure frequency, exposure duration, ingestion or contact rate, chemical concentration, body weight, and averaging time.

Intake can be described by the following general equation:

$$\text{Intake} = \frac{\text{Concentration} \times \text{Ingestion Rate} \times \text{Exposure Frequency} \times \text{Exposure Duration}}{\text{Body Weight} \times \text{Averaging Time}}$$

2.3.1 EXPOSURE ESTIMATION FOR NONCARCINOGENIC EFFECTS

The intake of chemicals evaluated for noncarcinogenic health effects is estimated over an averaging time dependent on the assessed toxic effect (i.e., health effect). This assessment evaluates chronic exposure to chemicals on the basis of systemic toxic effects and the estimated period of exposure. The averaging time for noncarcinogenic effects is equal to the exposure duration (i.e., 30 years) for this assessment.

2.3.2 EXPOSURE ESTIMATIONS FOR CARCINOGENIC EFFECTS

The intake of a chemical evaluated for carcinogenic health effects is referred to as the lifetime average chemical intake. The lifetime average chemical intake is calculated by prorating the total cumulative dose of the chemical over an averaging time of an entire life span (assumed to be 70 years) (EPA, 1989a and 1989b).

2.3.3 AVERAGE AND REASONABLE MAXIMUM EXPOSURE SCENARIOS

The need for remedial action at Superfund sites is based on an estimate of the RME or "reasonable maximum exposure." The RME is defined as the "highest exposure that is reasonably expected to occur at a site" (EPA, 1989a). The intent of the RME is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possibilities. Each exposure factor has a range of possible values. The following sections provide estimates of exposure factors for both the average and the RME scenarios.

2.4 CHEMICAL CONCENTRATIONS

The arithmetic mean chemical concentration is used to evaluate groundwater exposures for the average exposure scenario. The upper 95th percent confidence interval on the arithmetic mean of the data set is used for the RME scenario (EPA, 1989b). If a chemical is not detected in a particular sample, but is detected in other groundwater samples in the OU area, a value equal to 1/2 the detection limit is used to

estimate the exposure concentration, even though an estimated concentration was reported. The detection limit for all the COPCs was 2 μg/L. In cases where duplicate samples have been taken, the sample and duplicate results are averaged before summary statistics are calculated. The arithmetic mean, standard deviation, and upper 95 percent confidence interval on the arithmetic mean groundwater concentrations are summarized in Table 1. For the six COPCs for which no quantifiable levels were detected, only the value equal to 1/2 the detection limit is used as an estimate for the exposure concentration (1 μg/L in all cases), although this is clearly an overestimate. It is assumed that the concentration remains constant for the duration of the exposure period. Unvalidated historical data from wells in this area have shown significant fluctuation in contaminant concentration over the past twelve years. (See RI/FS Section 5.2)

2.5 EXPOSURE TO CHEMICALS IN HOUSEHOLD TAP WATER

Human exposure to contaminants in water used for domestic water supply can occur through three routes of exposure: ingestion, inhalation, and dermal absorption.

Ingestion. People can be directly exposed to contaminants in groundwater through the ingestion of tap water. The degree of exposure to contaminants through ingestion depends on the amount of water ingested on a daily basis. This assessment uses an ingestion intake value of 2 liters/day for both the average and RME scenario (EPA, 1989a).

The following equation is used for calculating daily chemical intake from ingestion of drinking water:

$$I = (CW \times IR \times EF \times ED) / (BW \times AT)$$

where:

I	=	Chemical intake (mg/kg body weight-day)
CW	=	Chemical concentration in water (mg/L)
IR	=	Ingestion rate (liters/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Table 1

CHEMICALS OF POTENTIAL CONCERN

Chemical	#Detect/ #Samples	Arithmetic Mean (µg/L)	Standard Deviation µg/L	Upper 95th Percentile Confidence Interval (RME) (µg/L	Maximum µg/L	Drinking Water Standards Maximum Contaminant Levels	
						US EPA (µg/L	California (µg/L
Tetrachloroethene (PCE)	26/26	12.96	10.27	16.4	36	5	5
Trichloroethene (TCE)	21/26	2.96	1.93	3.61	7	5	5
cis-1,2-Dichloroethene	18/26	1.31	0.68	1.54	3	70	6
1,1,1-Trichloroethane	1/26	1	NA	1	< 2	200	200
1,1-Dichloroethane	17/26	1	NA	1	< 2	NA	5
1,2-Dichloropropane	4/26	1	NA	1	< 2	5	5
Carbon Tetrachloride	2/26	1	NA	1	< 2	5	0.5
Chloroform	5/26	1	NA	1	< 2	100	NA
Methylene Chloride	8/26	1	NA	1	< 2	5	NA

Inhalation. Individuals can also be exposed to VOCs transferred from tap water to the air from showers, baths, toilets, dishwashers, washing machines, and cooking. Studies have suggested that exposure to volatile chemicals from inhalation can be as great as or greater than from ingestion alone. This assessment assumes that the dose from inhalation of VOCs is approximately equivalent to the dose from ingestion of 2 liters/day of the same water (EPA, 1989b).

Dermal. Another potential route of exposure associated with water use is dermal absorption of contaminants. Dermal absorption could occur during bathing, showering, food preparation, and washing dishes.

Skin is not very permeable and, therefore, is a relatively good lipoid barrier separating humans from their environment; however, some chemicals can be absorbed by the skin in sufficient quantities to produce systemic effects. Absorption of a chemical requires passage through the outer skin layer-the stratum corneum. Passage through this barrier is the rate limiting step in dermal absorption. It appears that, in general, toxicants move across the stratum corneum by passive diffusion following Fick's Law.

Specifically, however, many factors influence the absorption of chemicals across the skin layer. These include the health of the skin, the location of the area of skin exposed, hydration of the skin, time of exposure, molecular size of the chemical, lipid solubility, thickness of the skin, temperature, and the type of solvent the solute is dissolved in. Because dermal absorption is a complex process controlled by many factors, it is difficult to precisely predict exposures from this route.

Dermal absorption of volatile compounds in pure form or dilute solution has been observed and documented by several studies (Dutkiewitz and Tyros, 1967; Dutkiewitz and Tyros, 1968; Scheuplein and Blank, 1971; Scheuplein and Ross, 1974). There have been varying estimates on the amount of chemical intake that can result from dermal absorption of chemicals in water (Brown et al., 1984; and Levin et al., 1984). Cothern et al. (1985) suggest that intake through dermal absorption would normally be much less (by several orders of magnitude) than either the ingestion or inhalation routes in a household setting where exposure comes from the water supply. Estimation of household exposures by Foster and Chrostowski (1986) yielded similar results.

This risk assessment assumes that dermal absorption in the residential use setting is not a significant route of exposure.

Other. A lifetime average body weight of 70 kg was used to estimate both the average and RME scenarios (EPA, 1989b). For both exposure scenarios, an exposure frequency of 365 days per year is assumed. For both the average

exposure scenario and the RME scenario, it is assumed that a resident lives in their home for a duration of 30 years, the national upper-bound time (90th percentile) at one residence (EPA, 1989b).

3.0 TOXICITY ASSESSMENT

In this preliminary risk assessment, human health risks are evaluated in terms of noncarcinogenic and carcinogenic risks. Chemicals with carcinogenic risk frequently have noncarcinogenic effects, too.

3.1 NONCARCINOGENIC EFFECTS

Chemicals causing noncarcinogenic effects are believed to exhibit a level of exposure (from above zero to some finite value) that can be tolerated by the organism without causing an adverse health effect. Noncarcinogenic health effects include a variety of toxic effects on body systems, ranging from renal toxicity (toxicity to the kidney) to central nervous system disorders. It is believed that organisms might have protective mechanisms that must be overcome before a toxic endpoint (effect) is manifest. The toxicity of a chemical is assessed through a review of toxic effects noted in short-term (acute) animal studies, long-term (chronic) animal studies, and epidemiological investigations.

3.2 CARCINOGENIC EFFECTS

Carcinogenesis is generally thought to be a phenomenon for which risk evaluation based on presumption of a threshold is inappropriate. It is assumed that a smaller number of molecular events can evoke changes in a single cell that can eventually lead to cancer. This hypothesized mechanism for carcinogenesis is referred to as "nonthreshold" because there is assumed to be essentially no level of exposure to such a chemical that does not pose a finite probability, however small, of generating a carcinogenic response.

EPA has developed a carcinogen classification system (EPA, 1986a) that uses a weight-of-evidence approach to classify the likelihood of a chemical being a human carcinogen. This classification is listed in Table 2.

Table 2

EPA WEIGHT-OF-EVIDENCE CATEGORIES FOR POTENTIAL CARCINOGENS

EPA Category	Description of Group	Description of Evidence
Group A	Human Carcinogen	Sufficient evidence from epidemiology studies to support causal association between exposure and human cancer.
Group B1	Probable Human Carcinogen	Limited evidence of carcinogenicity in humans from epidemiology studies.
Group B2	Probable Human Carcinogen	Sufficient evidence of carcinogenicity in animals; inadequate evidence of carcinogenicity in humans.
Group C	Possible Human Carcinogen	Limited evidence of carcinogenicity in animals; no data for humans.
Group D	Not Classified	Inadequate evidence of carcinogenicity in animals.
Group E	No Evidence of Carcinogenicity in Humans	No evidence of carcinogenicity in at least two adequate animal tests or in both epidemiology and animal studies.

3.3 TOXICITY VALUES

Toxic response depends on the dose or concentration of the substance. Toxicity values are a quantitative expression of the dose-response relationship for a chemical. Toxicity values take the form of reference doses (RfDs) for noncarcinogenic effects and cancer slope factors (CSFs) for carcinogenic effects. Both RfDs and CSFs are specific to the exposure route.

The primary source of toxicity values used in this risk assessment is EPA's Integrated Risk Information System (IRIS) data base (EPA, 1993). The IRIS data base contains up-to-date health risk and EPA regulatory information. IRIS contains only those RfDs and CSFs that have been verified by EPA work groups and is considered by EPA to be the preferred source of toxicity information.

If a toxicity value is not available through IRIS, the next data source consulted is the most recently available Health Effects Assessment Summary Tables (HEAST) issued by EPA's Office of Research and Development. HEAST summarizes interim (and some verified) RfDs and CSFs.

3.3.1 Reference Dose

The toxicity value describing the dose-response relationship for noncarcinogenic effects is the RfD. The RfD is generally expressed in units of mg/kg-day. Inhalation RfDs may be expressed as either mg/kg-day or milligrams per cubic meter (mg/m^3) of air. Chronic RfDs are an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure to the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 1989a).

The RfDs used in this assessment are presented in Table 3. Inhalation RfDs are not available for some of the chemicals detected in groundwater in the OU area. In the absence of chemical-specific inhalation RfDs, oral RfDs are used.

Table 3

DOSE-RESPONSE VARIABLES FOR CHEMICALS OF CONCERN

Chemical	Systemic Toxicity (mg/kg-day)				Carcinogenic Potency (mg/kg-day)					
	Oral RfD	Source	Inhalation RfD	Source	Oral Slope Factor	Weight of [1] Evidence	Source	Inhalation Slope Factor	Weight of Evidence	Source
Tetrachloroethene (PCE)	0.01	IRIS	--	IRIS	0.055	B2/C	HEAST	0.002	B2/C	HEAST
Trichloroethene (TCE)	0.006	ECAO	--	IRIS	0.011	B2/C	HEAST	0.006	B2/C	HEAST
cis-1,2-Dichloroethene	0.01	HEAST	--	IRIS	--	D	IRIS	--	D	IRIS
1,1,1-Trichloroethane	0.09	HEAST	0.3	HEAST	--	D	IRIS	--	D	IRIS
1,1-Dichloroethane	0.1	HEAST	0.14	HEAST	--	C	IRIS	--	C	IRIS
1,2-Dichloropropane	--	IRIS	0.0012	IRIS	0.068	B2	HEAST	--	B2	HEAST
Carbon Tetrachloride	0.0007	IRIS	--	IRIS	0.13	B2	IRIS	0.091	B2	IRIS
Chloroform	0.01	IRIS	--	IRIS	0.0061	B2	IRIS	0.081	B2	IRIS
Methylene Chloride	0.06	IRIS	0.9	HEAST	0.0075	B2	IRIS	0.0016	B2	IRIS

IRIS = Integrated Risk Information System, EPA, 1993
HEAST = Health Effects Assessment Tables, EPA, 1993
ECAO = Environmental Criteria and Assessment Office, EPA, 1992
-- = Information not available

[1] Weight of Evidence Groups are listed in Table 2.

EPA's Environmental Criteria and Assessment Office has derived a provisional oral RfD of 0.006 (mg/kg-day) for trichloroethylene (EPA, 1992). This value has been used in this preliminary risk assessment.

3.3.2 Cancer Slope Factor

The dose-response relationship for carcinogens is expressed as a CSF. Generally, the slope factor is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The approach used to estimate the slope factor from animal studies or human data assumes a dose-response relationship with no threshold. Cancer risks estimated by this method produce an estimate that provides a rough but plausible upper limit of risk (i.e., it is not likely that the true risk would be much more than the estimated risk, but it could be considerably lower [EPA, 1989a]).

The cancer slope factors used in this risk assessment are presented in Table 3.

4.0 HEALTH RISK CHARACTERIZATION

This section presents an evaluation of the potential noncarcinogenic and carcinogenic risks to public health associated with the Newmark OU. The estimation of risk assumes that exposure remains constant over the exposure periods assessed (i.e., contaminant concentrations and intake levels are constant).

4.1 NONCARCINOGENIC RISKS

Noncarcinogenic risk is assessed by comparing the estimated daily intake of a chemical to its RfD. This comparison serves as a measure of the potential for noncarcinogenic health effects. The estimated intake of each chemical through an individual route of exposure is divided by its RfD. The resulting quotients are termed noncancer hazard quotients. When the hazard quotient exceeds one (i.e., intake exceeds RfD), there is potential for health concern (EPA, 1989a).

To assess the potential for noncarcinogenic effects posed by multiple chemicals, a "hazard index" approach has been adopted (EPA, 1989b). The method assumes dose additivity. Hazard quotients are summed to provide a hazard index. When the hazard index exceeds one, there is a potential for a cumulative health risk. If a single hazard quotient exceeds one, the hazard index will exceed one. The hazard index can also exceed one even if no single chemical intake exceeds its reference dose.

4.2 CARCINOGENIC RISKS

The potential for carcinogenic effects is evaluated by estimating excess lifetime cancer risk, which is the probability of developing cancer during one's lifetime over the background probability of developing cancer (i.e., if no exposure to site contaminants occurred). For example, a 1×10^{-6} excess lifetime cancer risk means that for every 1 million people exposed to the carcinogen throughout their lifetime (which is typically assumed to be 70 years) at the defined exposure conditions, the average incidence of cancer is increased by one extra case of cancer. Because of the methods used to estimate CSFs, the excess lifetime cancer risks estimated in this preliminary risk assessment should be regarded as upper bounds on the potential cancer risks rather than an accurate representation of the true cancer risk. The actual risk could be as low as zero.

Although synergistic or antagonistic interactions might occur among chemicals at the site, at this time there is insufficient information in the toxicological literature to predict quantitatively the effects of such interactions. Therefore, carcinogenic risks are treated in this preliminary risk assessment as additive within a route of exposure (EPA, 1986b).

The magnitude of cancer risk relative to Superfund site remediation goals in the National Contingency Plan ranges from 10^{-4} (one-in-ten-thousand) to 10^{-6} (one-in-one-million) depending on the site, proposed usage, and chemicals of concern (EPA, 1989a). Within this range, the level of risk which is considered to be acceptable at a specific site is a risk management decision and is decided on a case-specific basis. It is generally accepted that risks above this range require attention. The one-in-a-million level of risk (expressed as 1E-06) is often referred to as the *de minimis* level of risk; risks calculated below this range would not require attention. The 1E-06 risk level does not equate to an actual cancer incidence of one-in-

a-million. For substances that may cause cancer, the risk assessment process uses animal data to predict the probability of humans developing cancer over a 70-year lifetime. The numbers are given as upper bounds; the real risk is expected to be less. The one-in-a-million risk level is a theoretical prediction that no more than one person out of a million lifetimes would contract cancer due to an environmental exposure. By the way of comparison, the average person in the U.S. incurs a background risk of cancer (from all causes) of about one chance in four (0.25). Adding a risk of 0.000001 to a background risk of 0.25 is of little significance to any single individual. These small risk levels may be of concern only if the exposed population includes many millions of people.

4.3 ESTIMATED RISKS-QUANTITATIVE ASSESSMENT

Noncarcinogenic and carcinogenic risks have been calculated for each exposure pathway and for each COPC. Risks for individual chemicals are summed to estimate multichemical risks for each exposure pathway. A summary of estimated risks for residential exposure to groundwater from the Newmark OU area are presented in Tables 4 and 5 for noncarcinogenic effects and Tables 6 and 7 for carcinogenic risk.

The exposure scenario for the Newmark OU area assumes a future resident would come in contact with groundwater at the site through domestic use of tap water. Noncarcinogenic exposure levels do not exceed the RfDs for any individual COPC or for the sum of all nine COPCs. For average ingestion and inhalation exposure from the use of tap water, the sum of the noncancer hazard quotients are 0.1 for the ingestion (oral) route and 0.12 for the inhalation route. The total hazard index, based on an average exposure scenario, is 0.22. For reasonable maximum exposures from the use of tap water, the sum of the noncancer hazard quotients are 0.11 for the ingestion (oral) route and 0.14 for the inhalation route. The overall noncancer hazard index from RME to groundwater in the Newmark OU area is 0.25. The major chemical contributor to the overall noncancer hazard index, based on reasonable maximum ingestion and inhalation exposures, is PCE (0.094).

Table 4

SYSTEMIC TOXICITY SUMMARY
HAZARD INDEX CALCULATION FOR
REASONABLE MAXIMUM EXPOSURE (RME)
95% UPPER CONFIDENCE INTERVAL

Chemical	Concentration RME (µg/L)	Ingestion			Inhalation			Total Hazard Index
		Dose (mg/kg-day)	RfD (mg/kg-day)	Hazard Index	Dose (mg/kg-day)	RfD (mg/kg-day)	Hazard Index	
Tetrachloroethene (PCE)	16.40	4.7 E-4	0.01	0.047	4.7 E-4	--	(0.047)	0.094
Trichloroethene (TCE)	3.61	1.0 E-4	0.006	0.017	1.0 E-4	--	(0.017)	0.034
cis-1,2-Dichloroethene	1.54	4.4 E-5	0.01	0.004	4.4 E-5	--	(0.004)	0.008
1,1,1-Trichloroethane	1	2.9 E-5	0.09	0.0003	2.9 E-5	0.3	0.0001	0.0004
1,1-Dichloroethane	1	2.9 E-5	0.1	0.0003	2.9 E-5	0.14	0.0002	0.0005
1,2-Dichloropropane	1	2.9 E-5	--	--	2.9 E-5	0.0012	0.024	0.024
Carbon Tetrachloride	1	2.9 E-5	0.0007	0.041	2.9 E-5	--	(0.041)	0.082
Chloroform	1	2.9 E-5	0.01	0.003	2.9 E-5	--	(0.003)	0.006
Methylene Chloride	1	2.9 E-5	0.06	0.0005	2.9 E-5	0.9	0.00003	0.0005
TOTAL				0.113			0.136	0.249

Hazard Index = Exposure Dose (or Intake) / Reference Dose (or RfD)
In the absence of chemical-specific inhalation RfDs, oral RfDs are used.

$$\text{Intake (mg/kg-day)} = \frac{CW \times IR \times EF \times ED}{BW \times AT} = CW \times .02857 \text{ (liter/kg-day) (for non-carcinogens)}$$

where: CW = chemical concentration in water (mg/L)
IR = ingestion rate (= 2 liters/day, adult 90th percentile)
EF = exposure frequency (= 365 days/year, daily use by resident)
ED = exposure duration (= 30 years, national upper bound time (90th percentile) at one residence)
BW = body weight (= 70 kg/ adult average)
AT = averaging time (= 30 years x 365 days/year, for non-carcinogens)
AT = averaging time (= 70 years x 365 days/year, for carcinogens)

Table 5

SYSTEMIC TOXICITY SUMMARY
HAZARD INDEX CALCULATION FOR
ARITHMETIC MEAN

Chemical	Concentration RME (µg/L)	Ingestion			Inhalation			Total Hazard Index
		Dose (mg/kg-day)	RfD (mg/kg-day)	Hazard Index	Dose (mg/kg-day)	RfD (mg/kg-day)	Hazard Index	
Tetrachloroethene (PCE)	12.96	3.7 E-4	0.01	0.037	3.7 E-4	--	(0.037)	0.074
Trichloroethene (TCE)	2.96	8.5 E-5	0.006	0.014	8.5 E-5	--	(0.014)	0.028
cis-1,2-Dichloroethene	1.31	4.4 E-5	0.01	0.004	4.4 E-5	--	(0.004)	0.008
1,1,1-Trichloroethane	1	2.9 E-5	0.09	0.0003	2.9 E-5	0.3	0.0001	0.0004
1,1-Dichloroethane	1	2.9 E-5	0.1	0.0003	2.9 E-5	0.14	0.0002	0.0005
1,2-Dichloropropane	1	2.9 E-5	--	--	2.9 E-5	0.0012	0.024	0.024
Carbon Tetrachloride	1	2.9 E-5	0.0007	0.041	2.9 E-5	--	(0.041)	0.082
Chloroform	1	2.9 E-5	0.01	0.003	2.9 E-5	--	(0.003)	0.006
Methylene Chloride	1	2.9 E-5	0.06	0.0005	2.9 E-5	0.9	0.00003	0.0005
TOTAL				0.100			0.123	0.223

Hazard Index = Exposure Dose (or Intake) / Reference Dose (or RfD)
In the absence of chemical-specific inhalation RfDs, oral RfDs are used.

$$\text{Intake (mg/kg-day)} = \frac{CW \times IR \times EF \times ED}{BW \times AT} = CW \times .02857 \text{ (liter/kg-day) (for non-carcinogens)}$$

where: CW = chemical concentration in water (mg/L)
IR = ingestion rate (= 2 liters/day, adult 90th percentile)
EF = exposure frequency (= 365 days/year, daily use by resident)
ED = exposure duration (= 30 years, national upper bound time (90th percentile) at one residence)
BW = body weight (= 70 kg/ adult average)
AT = averaging time (= 30 years x 365 days/year, for non-carcinogens)
AT = averaging time (= 70 years x 365 days/year, for carcinogens)

Table 6

CARCINOGENIC RISK SUMMARY
REASONABLE MAXIMUM EXPOSURE (RME)
95% UPPER CONFIDENCE INTERVAL

Chemical	RME Concentration (µg/L)	Estimated Risk due to Ingestion	Estimated Risk due to Inhalation	Estimated Total Risk
Tetrachloroethene (PCE)	16.40	1.10 E-5	4.02 E-7	1.15 E-5
Trichloroethene (TCE)	3.61	4.86 E-7	2.65 E-7	7.51 E-7
cis-1,2-Dichloroethene	1.54	NA	NA	NA
1,1,1-Trichloroethane	1	NA	NA	NA
1,1-Dichloroethane	1	NA	NA	NA
1,2-Dichloropropane	1	8.33 E-7	NA	8.33 E-7
Carbon Tetrachloride	1	1.59 E-6	1.59 E-6	3.18 E-6
Chloroform	1	7.50 E-8	9.92 E-7	1.07 E-6
Methylene Chloride	1	9.18 E-8	1.96 E-8	1.11 E-7
TOTAL		1.41 E-5	3.27 E-6	1.74 E-5

Table 7

CARCINOGENIC RISK SUMMARY
ARITHMETIC MEAN

Chemical	Arithmetic Mean (μg/L)	Estimated Risk due to Ingestion	Estimated Risk due to Inhalation	Estimated Total Risk
Tetrachloroethene (PCE)	12.96	8.73 E-6	3.17 E-7	9.05 E-6
Trichloroethene (TCE)	2.96	3.99 E-7	2.18 E-7	6.16 E-7
cis-1,2-Dichloroethene	1.31	NA	NA	NA
1,1,1-Trichloroethane	1	NA	NA	NA
1,1-Dichloroethane	1	NA	NA	NA
1,2-Dichloropropane	1	8.33 E-7	NA	8.33 E-7
Carbon Tetrachloride	1	1.59 E-6	1.59 E-6	3.18 E-6
Chloroform	1	7.50 E-8	9.92 E-7	1.07 E-6
Methylene Chloride	1	9.18 E-8	1.96 E-8	1.11 E-7
TOTAL		1.17 E-5	3.14 E-6	1.49 E-5

Estimated excess lifetime cancer risks for average ingestion and inhalation exposures to tap water are 1.2 x 10^{-5} for the oral route (ingestion) and 3.1 x 10^{-6} for the inhalation route. The total estimated lifetime cancer risk for average residential exposure through domestic use of groundwater is 1.5 x 10^{-5}. The estimated excess lifetime cancer risk for RME from tap water is 1.4 x 10^{-5} for the oral route (ingestion) and 3.3 x 10^{-6} for the inhalation route. The total estimated lifetime cancer risk for reasonable maximum residential exposure through domestic use of groundwater is 1.7 x 10^{-5}. The major chemical contributor to the estimated lifetime cancer risk is PCE (1.2 x 10^{-5}).

Chemical-specific standards that define acceptable human health risk levels, such as MCLs, are also used in determining whether an exposure presents an unacceptable risk and whether remedial action is warranted (EPA, 1991b). The MCLs are the maximum permissible concentration of a chemical in water, which is delivered to any user of a public water system. Table 1 lists both state and federal MCLs for the COPCs. In the Newmark OU, PCE consistently exceeds the MCL, while the TCE concentration measured in groundwater occasionally exceeds the MCL. If PCE were treated to the MCL of 5 μg/L (ppb), and all other conditions and assumptions remained at the RME condition described above, the total estimated lifetime cancer risk for reasonable maximum residential exposure through domestic use of groundwater would be 9.4 x 10^{-6}. In that case, less than half of the total estimated risk would be contributed by PCE.

5.0 UNCERTAINTIES

The risk assessment is subject to uncertainty from a variety of sources. The main contributors to uncertainty include: sampling and analysis, fate and transport estimation, exposure estimation, and toxicological data.

Although risk assessment follows a formal scientific approach, making assumptions based on professional judgment is an inherent part of the process. Uncertainties inherent in the estimation of exposure and risks may act either to increase or decrease the identified risks, depending on the source of the uncertainty. This assessment is based upon the present understanding of the site characteristics and toxicology.

5.1 UNCERTAINTY IN EXPOSURE ASSESSMENT

Evaluation of uncertainty is an important component of the exposure assessment. Exposure point concentrations may be overestimated or underestimated depending on the conditions assumed and the actual conditions of exposure at the site.

Uncertainty in the quantitative estimates of chemical intakes is associated with each of the following assumptions under each scenario:

1. The arithmetic mean and the upper 95th percent confidence interval were used for exposure point concentrations and to represent the amount of chemical likely to be present in the groundwater. Actual chemical concentrations likely to be present in the groundwater may be more or less. Historic values have shown great variability.

2. Exposure scenarios represent idealized situations that may or may not represent actual, current, or future conditions.

3. The toxicity of potential COPC transformation products, whether having greater or less severe toxicity effects than chemicals discussed herein, are not accounted for in this evaluation.

4. The contamination in groundwater may not possess the mass or quantity of chemicals to provide the duration of exposure in each scenario. The actual exposure durations may be more or less depending on actual future conditions. The use of groundwater will depend on water quality and availability.

5. The assumption that the ingestion dose is equivalent to the inhalation dose for the shower, bathing, and other domestic use exposure may or may not be an overestimation. However, actual absorption through the oral route has been shown to be greater than through inhalation.

6. All COPCs except PCE and TCE were detected infrequently in samples from most wells in the Newmark OU, and concentrations were generally below levels that could be quantifiably measured. The convention (i.e., ½ the detection limit) used to estimate the intake of these compounds certainly overestimated the concentration used for the exposure assessment.

Therefore, all scenarios evaluated herein possibly overestimate actual exposure conditions that may exist in the future at the site. The exposure assessment presented in this report has attempted to provide best estimates rather than extreme estimates of exposure with respect to both the magnitude and duration of exposure at the site.

5.2 UNCERTAINTY IN TOXICITY INFORMATION

Varying degrees of uncertainty are associated with the toxicity values used to estimate potential health risks. Sources of uncertainty are due to the following:

1. The actual dose-response parameters and mathematical models used numerically estimated values based on the toxic effects of chemicals on laboratory animals.

2. Uncertainty is introduced by using the results of dose-response animal toxicity studies, for each chemical, to predict adverse health effects that may occur following human exposure to the low levels at the site. This uncertainty in using animal toxicity studies includes predicting the human health effects from short-term and long-term exposures. Margins of safety are inherent in the toxicity values derived for each chemical. These margins of safety are meant to be over-protective for the individual exposure at the site. Margins of safety for noncarcinogenic (threshold) chemicals are incorporated by the use of uncertainty factors when extrapolating the results of animal toxicity studies to predict the effects in humans. Margins of safety for carcinogenic (non-threshold) chemicals are incorporated by using the upper 95 percent confidence limit of the toxicity value. This value represents an upper 95 percent confidence limit on the probability of a response per unit intake of a chemical over a lifetime (i.e., there is only a 5 percent chance that the probability of a

response could be greater than the estimated value on the basis of the experimental data and model used to generate the toxicity value) (EPA, 1989a).

3. Toxicity values derived from the IRIS database system were accompanied with a qualitative description of their "strength of evidence" as determined by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) Workgroup; the corresponding confidence in each toxicity value was added to the uncertainty.

4. The toxicity assessment has been conducted in an effort to derive an appropriate numerical measure of adverse human toxic effects from the exposure to chemicals at the site. The current position of the State of California Department of Health Services (i.e., Cal/EPA cancer potency slope factors) has been considered in the derivation of toxicity values. The toxicity values are likely to be over-protective to the individual.

5. The use of oral RfDs for inhalation RfDs increases the estimate of risk due to differences in absorption rates.

5.3 UNCERTAINTY IN THE CHARACTERIZATION OF RISKS

The characterization of risks are not fully probabilistic estimates of the risk because no quantitative measure of uncertainty is associated with each numerical estimate. Rather, the risks are conditional estimates based on a considerable number of professional and subjective assumptions about the exposure and toxicity. The uncertainty about the numerical result is large (i.e., in the range of at least an order of magnitude or greater). It is more important to identify the key site-related variables and assumptions that contribute most to the uncertainty than to precisely quantify the degree of uncertainty in the risk characterization. The basic methodology used in this risk assessment was developed by EPA specifically for evaluation of risk at hazardous waste sites (EPA, 1989a). Overall, this methodology is conservative, which means that the true risks from the site are unlikely to be higher than the derived estimates, and are most likely lower.

6.0 ECOLOGICAL ASSESSMENT

This section addresses the potential ecological risks to flora and fauna in the area. It provides a qualitative evaluation of potential current and future risks represented by the present site conditions, assuming no remedial action is taken.

6.1 POTENTIAL RECEPTORS

The Newmark OU is zoned for commercial and industrial establishments. The surrounding area is a mixture of residential and commercial zoning. Although an extensive ecological survey has not been performed for the area, the presence of a significant wildlife population is not indicated. In addition, the developed condition of the site excludes the potential for significant natural vegetative cover.

6.2 POTENTIAL EXPOSURE PATHWAYS

The release pathway of primary concern at this site is contaminated groundwater. There is no information, at present, to indicate that this groundwater reaches the surface or that significant concentrations are discharged to a surface water source (i.e., canal, river, etc.).

6.3 CONCLUSIONS

Given the present developed condition of the site and the major exposure pathway consideration of contaminated groundwater, there is no expectation for significant impact to potential environmental receptors. Urbanization has already replaced habitat potential; therefore, no significant numbers of receptors, populations, or habitats appear to be present. There appears to be no apparent pathway for exposure to environmental receptors from contaminated groundwater. Also, there is no indication that future site plans would result in habitat creation or restoration and thereby recreate a potential for environmental receptors in the future.

BIBLIOGRAPHY

Brown, H.S., D.R. Bishop, and C.A. Rowan. 1984. The Role of Skin Absorption as a Route of Exposure for Volatile Organic Compound (VOCs) in Drinking Water. American Journal of Public Health, 74(5): 479-484.

Cothern, C.R., W.A. Coniglio, and W.L. Marcus. 1985. Techniques for the Assessment of Carcinogenic Risk to the U.S. Population Due to Exposure from Selected Volatile Organic Compounds from Drinking Water Via the Ingestion, Inhalation, and Dermal Absorption Routes. EPA 570/9-85-001.

Dutkiewitz, T. and H. Tyros. 1967. A Study of Skin Absorption of Ethylbenzene in Man. British Journal of Industrial Medicine, 24: 330-332.

———. 1968. Skin Absorption of Toluene, Styrene, and Xylene by Man. British Journal of Industrial Medicine, 25: 243.

Foster, S.A. and P.C. Chrostowski. 1986. Integrated Household Exposure Model for Use of Tap Water Contaminated with Volatile Organic Chemicals. Presented at the 79th Annual Meeting of the Air Pollution Control Association. June 22-27.

Levin, A., H.I. Maibach, and R.C. Wester. 1984. Assessment of Dermal Absorption of Contaminants in Drinking Water. Prepared for EPA, Office of Drinking Water, Draft Final Report. April.

Scheuplein, R.J. and I.H. Blank. 1971. Permeability of the Skin. Physiological Review, 51(4): 702-747.

Scheuplein, R.J. and L.W. Ross. 1974. Mechanism of Percutaneous Absorption V. Percutaneous Absorption of Solvent Deposited Solids. The Journal of Investigative Dermatology, 62: 353-360.

U.S. Environmental Protection Agency. 1986a. Quality Criteria for Water - 1986. EPA 440/5-86-001. Office of Water Regulations and Standards. May.

———. 1986b. Guidelines for the Health Risk Assessment of Chemical Mixtures. Federal Register 51: 34014-304025. September 24.

———. 1989a. Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A. Interim final. OSWER Directive 9285.701a. September.

U.S. Environmental Protection Agency. 1989b. Risk Assessment Guidance for Superfund, Human Health Risk Assessment. EPA Region IX Recommendations. December 15.

———. 1991a. National Guidelines for Organic Data Review. Contract Laboratory Program. June.

———. 1991b. Role of Baseline Risk Assessment in Superfund Remedy Selection Decisions. OSWER Directive 9355.0-30. April 22.

———. 1991c. Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Supplemental Guidance, "Standard Default Exposure Factors." Interim Final. OSWER Directive 9285.6-03. March 25.

———. 1991d. Health Effects Assessment Summary Tables - Annual FY 1991. OERR 9200.6-303(91-1). Office of Research and Development. January.

———. 1992. Environmental Criteria and Assessment Office. April 13.

———. 1993. Integrated Risk Information System. Chemical Files. EPA Integrated Risk Information System database, Office of Research and Development. Cincinnati, Ohio.